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16. ABSTRACT

This chapter addresses the physiological basis of differential plant sensitivity to pollutants. A generic model will be used to provide a conceptual framework for discussing differential plant sensitivity. After a brief description of the model, potential feedback control of physiological responses will be illustrated with examples from the literature. Plant responses to multiple pollutants, including the non-additive nature of stress response is briefly addressed.

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The Physiological Basis of Differential Plant Sensitivity to Changes in Atmospheric Quality

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INTRODUCTION

During the next several decades vegetation will continue to be exposed to a wide variety of pollutants, although the types of compounds, their concentrations and spatial patterns may change. Some are distributed globally, whereas others are distributed regionally or locally. These airborne pollutants can have either a direct impact on the plant foliage or act indirectly through deposition onto the soil and subsequent uptake by roots. These effects can range from subtle modifications of cellular biochemistry and whole-plant physiology (e.g., carbon allocation) to overt foliar injury and effects on plant growth, yield and/or reproduction.

Numerous studies have established that plants exhibit differential sensitivity to air pollutants (e.g., Tingey and Taylor 1982, Roose and Bradshaw 1982, Tingey and Olszyk 1985, Scholz et al. 1989). Various hypotheses have been proposed to explain differential pollutant sensitivity, however, in most cases the mechanism(s) of the differential response is not known. Nevertheless, the underlying source of variation in plant response is disparate physiological states among the various individuals. These disparate states are important because they are due, at least in part, to genetic variation.

Pollutant resistance is often a quantitative trait involving numerous genes (Pitelka 1988). Heritability for resistance tends to be high and the genes for resistance often work in an additive manner (Roose and Bradshaw 1982, Dragoescu et al. 1988, Houston and Stairs 1973, Karnosky 1977, Taylor 1978, Butler et al. 1979, Hanson et al. 1976). Resistance enables the plant to survive, grow, and reproduce in the presence of a particular pollutant (Pitelka 1988). Populations possessing latent variability for pollutant resistance can evolve resistant populations if exposed to high concentrations of pollutant for sufficient time (Pitelka 1988). In contrast, populations lacking such variation cannot become resistant and may decline under pollutant stress.

Pollutant stresses can have a profound impact on genetic diversity even though stressed individuals rarely die as a direct result of current ambient pollutant levels. Pollutant stresses can reduce growth, reproductive fecundity, and competitive status in sensitive individuals, without causing death. The population consequence of reduced fecundity would be a shift from the sensitive to resistant types in future generations. In this manner, pollutant stresses can impact evolutionary processes occurring in plant communities. The overall result is a reduction in genetic diversity with a concomitant reduction in tolerance to other stresses (Karnosky et al. 1989).

Other chapters will specifically address population level responses to changes in atmospheric quality. This chapter addresses the physiological basis of differential plant sensitivity to pollutants. A generic model will be used to provide a conceptual framework for discussing differential plant sensitivity. After a brief description of the model, potential feedback control of physiological responses will be illustrated with examples from the literature. Plant responses to multiple pollutants, including the non-additive nature of stress response is briefly addressed.

FACTORS AFFECTING PLANT RESPONSE

The conceptual model (described below, Fig. 1) is not specific for any pollutant or pollutant type (i.e., gaseous or wet deposition), but provides a conceptual framework to understand the influence of genetic control on differential plant sensitivity. The model proposes a common progression of events, based on a multiplicity of interactive physiological mechanisms.

Plant response to a pollutant is the consequence of the interaction among various avoidance and tolerance mechanisms (Ariens et al. 1976, Tingey and Taylor 1982) (Fig. 1). Avoidance factors influence both the amount and the rate at which the pollutant or its metabolites reach the target site; tolerance

factors operate through processes that repair or compensate for the stress. Various feedback mechanisms can also influence plant response. The specific mechanisms for avoidance and tolerance may be broad based, i.e., responding to all stresses or it may be a stress-specific reaction. Although the target site for each stress is unique, it is the interactive balance among avoidance and tolerance processes that controls plant response to any stress.

AVOIDANCE FACTORS

Avoidance factors influence the amount/rate of the pollutant reaching the target site and consist of two phases: pollutant uptake and pollutant transport/metabolism (Fig. 1):

Pollutant Uptake

A pollutant stress response can occur only after the stress factor enters the plant. The uptake of airborne pollutants is dependent upon: (1) The concentration of the pollutant in the surrounding air; (2) the stomatal and cuticular conductances of the pollutant; and (3) its solubility. Stomata are the principal plant barrier influencing the uptake of gaseous pollutants. Cuticular characteristics are important for controlling the uptake of wet and dry deposited pollutants.

During the day, stomatal factors are most important while at night cuticular characteristics predominate. However, for gases such as O_3, cuticular conductance is several orders of magnitude smaller than stomatal conductance (Kerstiens and Lendzian 1989).

Pollutant transport/metabolism: These processes include: (1) absorption and distribution over the tissues, (2) transport (via membranes) and binding to the tissue components and; (3) metabolic alteration of the toxicant including changes in its chemical properties/sequestering, and if possible, excretion.

TARGET SITE INTERACTION

The pollutant target site is likely specific for a given class of compounds. If the pollutant reaches the target site in sufficient quantity, it will elicit a plant response. The concentration of the toxicant attained in the target tissue and the specific characteristics of the target site influence the magnitude of the resultant biological stress.

TOLERANCE FACTORS

Tolerance (repair and compensation) mechanisms lead to a new metabolic state following a target interaction and can be separated into two categories; reserve capacity, and modification

of gene expression (Queiroz 1988) (Fig. 1).

Reserve Capacity

The plant utilizes only a fraction of its existing metabolic capacity under normal conditions, i.e., enzyme capacity generally exceeds demand (Queiroz 1988). Consequently the initial response to a pollutant episode or increased pollutant stress is achieved using existing enzyme capacity with little additional energy cost.

Modification of Gene Expression

If there is insufficient enzyme capacity to achieve a new metabolic homeostasis, then de novo enzyme synthesis may occur (Queiroz 1988). This includes increased enzyme concentrations or the synthesis of new isoenzymes that may be more efficient. This pathway, which involves the synthesis of new protein, is energetically expensive. The pathway also includes the possibility of post-translational enzyme modification such as covalent modifications of enzymes by phosphorylation/dephosphorylation and synthesis of secondary metabolites.

Consequently, the initial response to a pollutant episode or increased pollutant level is achieved by utilizing the currently available enzyme capacity, with further response resulting in de novo enzyme synthesis. If repair is complete there will be no detectable effect except an increase in energy for repair and

maintenance of the metabolic pools. The magnitude of injury reflects the extent to which the metabolic machinery is able to repair or compensate for the stress. It is probable that both paths act in concert when the plant is exposed to a pollutant. If the plant cannot reestablish metabolic homeostasis, it will be destabilized (Fig. 2).

Repair/compensation may not be achieved if the pollutant impact disrupts the metabolic process(es) before the tolerance mechanisms are operative. This inhibition of metabolic adjustments offers an explanation for the synergistic effects from combinations of NO_2 and SO_2. Plants are able to tolerate low levels of NO_2 and it may even be beneficial (Wellburn 1982). This tolerance is accomplished by the enzymatic reduction of NO_3^- and NO_2^- to NH_4^+ (Zeevaart 1976). However, SO_2 inhibits the induction of additional nitrite reductase and the bioenergetic levels are significantly lower in the plants receiving the combined exposure. These factors combine to cause the injury.

The conceptual model (Fig. 1) proposes a progression of events, based on a multiplicity of interactive physiological mechanisms, with multiple points for environmental/metabolic control. Consequently, any one or a combination of several events may contribute to the observed differential response. This has led to the suggestion that different mechanisms of resistance may be involved among various species and pollutants.

However, from a conceptual viewpoint this is unlikely. A more reliable hypothesis is that the relative importance of avoidance and tolerance mechanisms varies among species/genotypes. For example, pollutant exclusion (avoidance) may be the dominant mechanism in one genotype while a modification of gene expression (tolerance) is more important in another. In either case, the primary basis of the differential sensitivity conforms to the general model of plant response to pollutants. Examples will be drawn from the literature to illustrate the various components of the model (Fig. 1), and show how they influence the response to various pollutants.

MECHANISMS OF RESPONSE

AVOIDANCE MECHANISMS

Pollutant Uptake

Phytotoxicity results from biochemical processes that occur within the plant cell or on its surface. Consequently the ease with which the pollutant moves from the ambient air into the plant is a key factor controlling plant response (Fig. 1). Gaseous pollutant uptake is affected by factors including patterns of stomatal opening and closing, stomatal frequency, morphological characteristics, and cuticle composition and structure. Using a combination of either physical or chemical

processes, the pollutant stress can be either partially or completely excluded from the plant (Levitt 1972). Resistant cultivars of Allium cepa L. displayed rapid stomatal closure when the plants were exposed to O_3, which resulted in complete stress avoidance (Engle and Gabelman 1966). However, partial stress avoidance is more common (Tingey and Taylor 1982). For example, in Petunia hybrida Vilm. O_3 injury was positively correlated with leaf conductance (Thorne and Hanson 1976).

Ozone and SO_2 can alter normal patterns of stomatal opening and closing (Winner et al. 1988), thereby affecting further pollutant uptake. Pollutant-induced avoidance may result from a direct impact of the pollutant on the guard cells or may act indirectly through changes in the carboxylation capacity and a subsequent change in the internal CO_2 concentration. Direct stomatal closure in response to SO_2 may result from guard cell death and subsequent loss of turgor (Black and Black 1979a and b). Indirect stomatal closure has been observed in Raphanus sativus L. and other species exposed to SO_2 (400 ppb) (Winner et al. 1988, Alscher et al. 1987). In these cases, photosynthesis decreased prior to a decrease in conductance, and stomatal closure may have resulted from an increase in internal CO_2 concentration. The mechanisms of stomatal closure may vary depending on pollutant concentration. Feedback mechanisms such as stomatal closure ultimately act to exclude further pollutant uptake, effectively increasing the threshold concentration

necessary for response.

The stomatal control of gas exchange is under both intrinsic and environmental control (Larcher 1975, Nobel 1974). Some species have intrinsically higher O_3 uptake rates than others. For example, most herbaceous plants have a higher uptake rate than woody species; uptake is highly correlated with the transpiration rate (Thorne and Hanson 1972). While broad-based generalizations are difficult to make, species exhibiting high stomatal conductances tend to be more susceptible to gaseous pollutant uptake (Reich 1987).

The intrinsic (gene) control of pollutant uptake can be overridden by environmental or edaphic factors (e.g., Tingey and Taylor 1982). For example, drought stress can override intrinsic plant sensitivity to O_3. Tingey et al. (1982) reported a decrease in O_3 sensitivity in water stressed Phaseolus vulgaris L. which was correlated with decreased leaf conductance. The response was related to decreased O_3 uptake in water stressed plants and was reversible when water stress was removed. Other environmental factors such as humidity can also override intrinsic factors affecting pollutant uptake (Black and Unsworth 1980, Winner et al. 1988, McLaughlin and Taylor 1981). McLaughlin and Taylor (1981) found that foliar O_3 and SO_2 uptake was increased 2 to four-fold by an increase in relative humidity from 35 to 75%. The mechanism underlying these differences was

related to a change in internal leaf conductance rather than an effect on the stomata.

Genetic factors controlling cuticular composition and structure can also modify plant response to pollutants. Raddi and Rinallo (1989) correlated the pollutant sensitivity of two provenances of Abies alba Mill. to epistomatal wax characteristics. The tolerant provenance maintained fibrillar waxes longer with age and pollutant exposure. Tolerant trees growing in both polluted and non-polluted areas maintained their fibrillar integrity, suggesting that there was a genetic basis for their relative sensitivity. While other morpholcgical factors could not be eliminated, the results suggest that pollutant sensitivity was related to cuticular properties and that amorphous plug formation may adversely effect gas exchange capabilities. In another study, O_3 exposure altered epicuticular wax structure in Picea abies (L.) Karst. (Barnes et al. 1988). These workers found a change from the crystalline arrangement of fine tubes to an amorphous layer that ultimately occluded the stomata in the trees exposed to O_3. This change is typically associated with needle aging, and O_3 was shown to accelerate the process. Stomatal occlusion may limit further uptake of pollutants, however, CO_2 diffusion may also be limited and could contribute to premature needle shed (Sauter and Voss 1986). In addition, stomatal occlusion may result in greater photo-oxidation and increased leaf temperatures due to a reduction in

evaporative cooling. Barnes and Davison (1988) found that O_3 exposure increased cuticular transpiration in Norway spruce, which may predispose trees to winter desiccation. Genotypic variation in epicuticular wax degradation appears to be high, suggesting genetic control of composition may influence pollutant response.

Genotypic differences in cuticular composition or thickness may be part of an avoidance mechanism for UV-B. Enhanced UV-B radiation significantly increased cuticular waxes through accelerated wax biosynthesis (Steinmüller and Tevini 1986). Although most cuticular waxes do not absorb within the visible and near UV-wave lengths, the structural arrangement of the waxes on the leaf surfaces appears to enhance light reflection and scattering (Steinmüller and Tevini 1986). One could postulate O_3-induced changes in surface wax structure (Barnes et al. 1988) could also alter its UV reflectance and consequently influence the avoidance mechanism.

Intrinsic and environmental factors affecting leaf conductance (stomatal and cuticular) affect the rate at which pollutants diffuse into the leaf interior. This relationship has obvious consequences for the concentration of the pollutant or its metabolites in the cell, which influences the magnitude of plant response to the pollutant. However, in most cases, knowledge of pollutant uptake rate alone is insufficient to

predict plant sensitivity (e.g., Tingey and Taylor 1982, Tingey and Olszyk 1985) because of the range of interactions which influence plant response. Pollutant uptake is only one of several important factors that control pollutant sensitivity (Fig. 1).

Transport to Target Site

Once a pollutant comes in contact with the leaf interior, factors such as solubility, absorption rate, transport, metabolism and detoxification processes influence the pollutant concentration/amount that reaches the target site.

Foliar sensitivity to O_3 has been associated with ascorbic acid levels; plants with high levels are less sensitive than plants with low levels (e.g., Hanson et al. 1971, Lee et al. 1984, Tanaka et al. 1985). Recent calculations by Chameides (1989) have suggested that a major fraction of the O_3 diffusing through the stomata could be detoxified via reaction with ascorbic acid in the extracellular H_2O. This possible mechanism for reducing the O_3 concentration before it reaches the target site is supported by the observation that O_3 exposures initially reduce ascorbic acid concentrations (e.g., Tanaka et al. 1985). Subsequently, there is an increase in the ascorbic acid concentration (to 1mM) and cationic peroxidase activity in the extracellular water of leaves (e.g., Lee et al. 1984, Castillo and Greppin 1986).

When SO_2 dissolves into H_2O in and on the cell, it is rapidly hydrated, forming HSO_3^{-1} and SO_3^{-2} (Peiser and Yang 1985). These compounds are substantially more toxic than SO_4^{-2} and have both been implicated in SO_2 toxicity (Thomas 1961). In Lycorpersicon esculentum Mill. foliar SO_3^{-2} levels increased with SO_2 exposure, and tissue SO_3^{-2} concentrations were highly correlated with foliar injury (Howe and Woltz 1981).

The plasma membrane was found to be a barrier to the uptake and transport of both SO_2 and SO_3^{-2} in studies on the inheritance of SO_2 resistance in crosses and inbred lines of Cucumis sativus L. (Bressan et al. 1981). The authors suggested that this membrane barrier contributed to differential SO_2 sensitivity. They supported their conclusion by the observation that the relative resistance to SO_2 or HSO_3^{-1} injury was the same for a variety of other stresses likely to affect membrane properties.

Detoxification systems capable of protecting sensitive tissues from pollutants and their derivatives are common in plants and are subject to genetic control (avoidance mechanisms). The oxidation of SO_3^{-2} to SO_4^{-2} has been proposed as a potential detoxification mechanism for SO_2. Exogenous SO_3^{-2} was metabolized more rapidly in Glycine max (L.) Merr. cultivars resistant to foliar SO_2 injury than in sensitive cultivars (Miller and Xerikos 1979). Kondo et al. (1980) reported that SO_2

tolerance in various species was correlated with sulfite oxidase activity. Ayazloo et al. (1982) found that a sensitive genotype of Lolium, S23, had a higher proportion of inorganic S as SO_3^{-2} than the tolerant genotypes did after both light and dark incubations in SO_3^{-2} solutions.

The photoreduction of SO_3^{-2} to H_2S and subsequent volatilization is another possible detoxification mechanism (Rennenberg 1984). Dodrill (1976) suggested that the emission of H_2S was a method of detoxifying SO_2, since H_2S emissions were stimulated at lower SO_2 concentrations than would cause foliar injury. Plants exposed to SO_2 emit H_2S in the light (DeCormis 1968, Silvius et al. 1976, Hällgren and Fredriksson 1982). Hydrogen sulfide was detected within 15 to 30 minutes after the start of exposure, with emissions reaching a maximum within 45 minutes (DeCormis 1968, Taylor and Tingey 1983). An uptake rate of at least 48 μmol SO_2 m^{-2} over 15 min was required to stimulate emissions, suggesting that the cellular concentration had to exceed a threshold before emissions occurred (Taylor and Tingey 1983). However, differential emission rates ("detoxification") were not correlated with ecotypic differences in SO_2 sensitivity (Taylor et al. 1986).

Ozone and SO_2 give rise to free radicals and H_2O_2 within plant cells (e.g., Heath 1980, Tingey and Taylor 1982, Alscher and Amthor 1988). Biological systems have evolved protective

scavenging or buffering systems such as superoxide dismutase (SOD), catalase (CAT), peroxidases, ascorbate, and α-tocopherol in response to free radicals (e.g., Heath 1980, Alscher and Amthor 1988). Superoxide dismutase converts the superoxide anion to H_2O_2 which is subsequently decomposed to H_2O and O_2 by catalase. The importance of free-radical scavenging mechanisms in aerobic metabolism (Fridovich 1978, Salin 1988) suggests that they are important processes controlling the amount of O_3 reaching the cellular target site. The rates of pollutant absorption appear to be more important than the total absorbed dose (Bennett et al. 1984). Healthy leaf mesophyll tissue can quantitatively absorb subinjurious concentrations of O_3. If the redox enzymes and other detoxifying processes are functioning, limited protection may be expected if saturation conditions are not exceeded.

The pools of SOD and CAT are under genetic and environmental control (Scandalios 1987). Ethylene diurea (EDU), a chemical that reduces O_3 injury, induces higher tissue levels of SOD and CAT in Phaseolus vulgaris L. leaves; these elevated levels were always correlated with reduced foliar injury (Lee and Bennett 1982, Bennett et al. 1984). However this clear association between elevated levels of scavenging enzymes and foliar injury is not found in all Phaseolus vulgaris L. cultivars. For example, O_3 exposure increased SOD activity in Phaseolus vulgaris L. leaves, and in some cultivars lipid soluble antioxidants

(McKersie et al. 1982). However, these increases in enzyme activity were not always the physiological basis of differential cultivar sensitivity. Chanway and Runeckles (1984a) found that EDU did induce protection from O_3 injury but SOD levels were not increased in their cultivars. However, SOD levels did increase simultaneously with the appearance of visible foliar injury (Chanway and Runeckles 1984b).

The apparent differential importance of SOD and CAT among Phaseolus vulgaris L. cultivars discussed above does not invalidate the role of SOD as an avoidance mechanism. The conceptual model indicates that there are several control points (both avoidance and tolerance) that are involved in differential sensitivity. The various Phaseolus vulgaris L. studies failed to provide a broad scale assessment of the various avoidance and tolerance factors that controlled plant response. It is likely that among various cultivars the several avoidance and tolerance mechanisms were of differential importance.

TOLERANCE MECHANISMS

To understand the effects of a pollutant, it is more important to understand the processes driving the metabolic adjustments rather than the metabolic fate of specific pollutant molecules. Plant metabolism is functionally compartmentalized with interconnected metabolic pathways that are optimized by

feedback loops. Balance between the metabolic pathways is continuously controlled and adjusted in response to environmental changes. The responses to environmental change involve coordinated readjustments in the plant's metabolic paths rather than a few "specific" reactions (Queiroz 1988).

Plant response to stress is a function of the interaction between the frequency or intensity of the stress and the rate at which the plant allocates resources for maintenance, repair and defense. Plants allocate resources in a manner which overcomes the resource deficiency that most limits plant growth. Allocation occurs in a direction that allows the plant to deal with or compensate for the stress. Such a result has the obvious evolutionary advantage of bringing the plant into balance with its biotic and abiotic environment.

Reserve Capacity

Repair and compensatory mechanisms that lead to a new metabolic state following the target interaction can utilize the existing capacity of the plant, without a modification of gene expression (Queiroz 1988). Wellburn et al. (1972) reported that the SO_2-induced swelling of thylakoids was reversible if polluted air was replaced by clean air. In Chlorella, O_3 increased potassium efflux, however, within 2-3 minutes after ozonation stopped the normal efflux rate was reestablished suggesting an active repair process (Heath et al. 1974). The rate of recovery

was too rapid to be attributed to new protein synthesis. In Phaseolus vulgaris L. leaves, the membrane repair rate was enhanced by continuous light or glucose supplements and retarded by low temperatures or darkness following exposure (Sutton and Ting 1977a,b). One early symptom of acute pollutant injury is the appearance of water-soaked interveinal areas on leaves. If these areas are not subjected to extensive drying by either wind or sunshine, H_2O may be reabsorbed into the cells, and thus result in little or no visual injury; the initial stages of injury are therefore reversible.

Ozone has been found to increase ATP and total adenylate contents (Pell 1974), and increase respiration rates in plants (Amthor 1988, Amthor and Cumming 1988, Barnes 1972, Skärby et al. 1987). Skärby et al. (1987) found that accumulated respiration was 60% higher in O_3-exposed shoots than in controls of 20-year-old Pinus sylvestris L.. Amthor (1988) and Amthor and Cumming (1987) mathematically partitioned respiration into growth and maintenance components in Phaseolus vulgaris L. exposed to O_3. They found that growth respiration was unaffected by O_3 while that due to maintenance was increased by as much as 25%. If sufficient carbon substrates are available, the reserve capacity may be sufficient to accommodate the increased maintenance and repair costs associated with O_3 stress.

Another example of homeostatic readjustment via reserve capacity can be found in the glutathione system, which prevents oxidative breakdown of unsaturated lipids and biomembranes, and maintains proteins, cysteine and homocysteine in a reduced state (i.e., metabolically active) (Leshem 1988, Rennenberg 1982). This protection is mediated by the oxidation of the thiol groups on the tripeptide to form the glutathione dimer conjugate (GSSG). In chloroplasts, this oxidation is mediated by the ascorbate-dehydroascorbate cycle (Leshem 1988, Salim 1988); glutathione can also be directly oxidized by oxidants such as O_3 (Mudd 1982). Glutathione reductase reduces GSSG to glutathione using NADPH for the reducing equivalents (Leshem 1988, Rennenberg 1982). The reduction of GSSG is important to maintain metabolism as GSSG can inactivate light-activated enzymes in the Calvin cycle (Rennenberg 1982). The reduction of GSSG to glutathione via glutathione reductase would be expected to stimulate the pentose phosphate pathway (Mudd 1982). Ozone has been shown to stimulate the activity of glucose-6-phosphate dehydrogenase suggesting the pentose phosphate pathway was stimulated (Tingey et al. 1976). The increased enzyme occurred earlier in the sensitive cultivar than the tolerant one, possibly as a result of greater glutathione oxidation.

Plant protective mechanisms to UV may be grouped into three main classes: (1) UV damage is repaired or its effects are negated; (2) the amount of UV radiation reaching the target site

is reduced; and (3) plant response minimizes the effects of the UV stress (Beggs et al. 1986). An example of a repair process is the photoreactivation of UV stress. The inhibitory effect of UV on leaf enlargement is less if the plants are placed in light immediately following UV exposure than if they are placed in the dark (Beggs et al. 1986). UV stress, as measured by the UV-induction of coumestrol, was reversible if the plants were placed in white light but not blue light (Beggs et al. 1985). Sunlight counteracts the UV-inhibition of phytochrome mediated anthocyanin formation (Beggs et al. 1986). In Glycine max (L.) Merr., UV-B inhibited photosynthesis only in leaves that expanded and were irradiated under a low PPFD indicating that a high PPFD during leaf expansion or irradiation was effective in minimizing the detrimental effects of UV-B (Mirecki and Teramura 1984). Warner and Caldwell (1983) reported the relative magnitude of photosynthesis inhibition was similar under either light-limited or light-saturated conditions. However, under the low light growth conditions, UV-B treatment reduced chlorophyll content, while no effect was observed under the light-saturated treatment.

Modification of Gene Expression

Under environmental stress, cells can rapidly respond in a specific manner by selectively increasing or decreasing the expression of specific genes (Sachs and Ho 1986). Genes whose expression is increased during times of stress presumably are critical to the accommodation of the organism to adverse

conditions (Matters and Scandalios 1986). The variety of proteins and mRNAs which are synthesized during stress indicates no exclusive simple group of "stress proteins" per se, although several genes, such as those encoding the heat shock proteins, are commonly expressed under more than one stress condition (Matters and Scandalios 1986). The function of many of the stress proteins is unknown, but the highly reproducible and specific nature of stress proteins indicates a specific role in plant metabolic systems. Other compounds, such as stress ethylene, are produced under a variety of stresses and may mediate plant processes (Gunderson and Taylor 1988).

There is substantial evidence suggesting that increased oxidative stress produced by environmental factors can alter superoxide dismutase (SOD) and catalase (CAT) gene expression in many organisms (Scandalios 1987). Oxidative stress induces specific isozymes of SOD, and enhances the expression of both SOD and CAT genes. A variety of environmental factors can create conditions of intracellular oxidative stress (redox-active compounds like paraquat, elevated O_2, hyperbaric O_2, air pollutants and increased temperature) which result in these metabolic readjustments.

In animals, metal-binding proteins (metallothionein) have been identified, isolated, and implicated in the detoxification of and protection from metal toxicity as well as the storage and

transport of metals (Cherian and Goyer 1978). Plants exposed to heavy metals such as Cd or Zn produce "metallothionein-like" proteins which bind heavy metals (Blakeley et al. 1986, Grünhage et al. 1985, Rauser 1984, Wagner and Trotter 1982). These proteins, rich in cysteine, are found in both the roots and leaves. Heavy metal complexing peptides are also formed (Grill et al. 1985, Robinson and Jackson 1986). The observations that the metal-binding proteins are metal inducible has led to the conclusion that these compounds function as a protective system against metal toxicity (Robinson and Jackson 1986). The contention is supported by the observation that metal tolerant plants produce higher than normal levels of the metal-binding proteins. However, metal sensitive and tolerant plants both produce the protein. In some genotypes, metal binding proteins appear to be important avoidance/tolerance mechanisms (Rauser and Curvetto 1980, Robinson and Jackson 1986).

Plant protective mechanisms to ultraviolet light can influence the amount of UV radiation reaching the target site, which is an example of feedback control increasing stress avoidance. For example, UV induces the formation of flavonoids, anthocyanins and phenols in plant tissue (Caldwell et al. 1983, Beggs et al. 1986, Robberecht and Caldwell 1986). The flavonoids and related phenols are wave-length selective filters for UV which allow the penetration of visible radiation but remove much of the UV radiation. Flavonoid synthesis is apparently mediated

via UV-induced pyrimidine dimers (Beggs et al. 1985). UV treatment causes the coordinated induction of enzymes in the flavonoid biosynthetic path, including, phenylalanine ammonia-lyase, 4-coumarate:CoA ligase, chalcone synthase and UDP-apiose synthase (Kuhn et al. 1984). Using cDNA probes, it was found that the UV treatment increased the levels of the mRNA encoding these enzymes and that this increased the transcription rates of the genes (Kuhn et al. 1984, Chappell and Hahlbrock 1984). These compounds accumulate in the epidermis of plants exposed to UV and can significantly affect the amount of UV radiation reaching the mesophyll tissue. A UV sensitive species, Rumex patientia L., has a higher epidermal transmission than R. obtusifolius L. (Robberecht and Caldwell 1986).

Ozone exposure also increases the activity of phenylalanine ammonia-lyase and induces the formation of phenols and anthocyanins in leaf tissue (e.g., Tingey et al. 1976, Koukol and Dugger 1967, Howell and Kremer 1973). The similar effect of O_3 and UV light on these metabolites leads to the suggestion that the O_3-induced increase in UV-B absorbing metabolites could provide a partial protection from UV-B. However, it is unlikely that UV-induced increase in phenols and anthocyanins will alter O_3 sensitivity as there is not evidence that they are involved in either O_3 tolerance or avoidance mechanisms.

Foliar received NO_2 can induce the formation of nitrate reductase in plant foliage (Zeevaart 1974, Wellburn 1982, Winsle et al. 1987, Rowland 1986, Norby et al. 1989). Nitrogen dioxide reacts with extracellular H_2O and results in the formation of NO_3^- and NO_2^-. In addition, Lindberg et al. (1986) has shown that dry deposition of NO_3^- (primarily as HNO_3 vapor) is an extremely important source of anthropogenic N input into forested ecosystems. Nitrate reductase enzymes reduce NO_3^- to NH_4^+ which is subsequently metabolized into amino acids and proteins (Zeevaart 1976, Rogers et al. 1979, Rowland et al. 1987). This inducible enzyme system is an example of stressor-induced changes in gene expression. The consequence of the altered gene expression is enzyme induction with the subsequent metabolism of the stressor and its sequestering amino acids and protein.

Various inducible systems appear to be important in pollutant stress response (i.e., "stress proteins" or "stress metabolites"). At this time, no single factor (initiator) seems to be responsible for inducing "stress proteins" or "stress metabolites". Air pollutants stimulate the formation of stress ethylene, as do other abiotic and biotic stresses (e.g., Abeles 1973, Bressan et al. 1979, Tingey 1980). This hormone may function as a signal that triggers changes in gene expression during stress. An increase in abnormal or denatured proteins occurs under heat shock, which may serve as the trigger (Matters and Scandalios 1986). The altered proteins are preferentially

degraded by cellular proteases, causing the build-up of the heat shock-activating factor and the induction of heat shock gene expression.

INTERACTIONS OF CHANGES IN ATMOSPHERIC QUALITY

Although it is important to understand the basic mechanisms influencing plant response, it is equally important to consider how the basic response mechanism will be influenced by changes in other environmental factors such as altered rainfall (i.e., drought) or elevated CO_2 concentrations, as the various stress factors seldom occur individually. There have been extensive studies on the effects of individual factors on the mechanism of plant response, but there has been little concern for the interactive effects of several environmental stress factors.

Johnson (1989) developed a conceptual model for evaluating factors involved in forest decline in northeastern United States (Fig. 2). This model, developed from concepts described earlier by Manion (1981), is based on the available energy for avoidance, repair and compensation processes. As such it is conceptually similar to our model for differential sensitivity (Fig. 1). At either the cellular or the individual level, a major factor in determining if repair/compensation occurs and a new stable metabolic state is reached is the available energy (carbon). In Johnson's model, predisposing factors reduce the amount of carbon

substrate available for defense (avoidance) and repair/compensation, but not to the extent that a response threshold level is reached. An inciting stress (either atmospheric pollutant or abiotic/biotic stress) shifts the response below a threshold level, resulting in the appearance of stress symptoms. If the inciting stress is sufficient to reduce available carbon for repair to a level below the critical level, then the decline phase may directly result. If the stress is episodic, the plant may recover. However, if a secondary episodic stress (the same or different) occurs, it could delay the time to recovery or sufficiently reduce the available energy to shift the response below the critical level, resulting in decline. Each phase is a continuous process which involves metabolic readjustments, the magnitude of which are determined by the availability of carbon substrates. The decline stage in the Johnson model is analogous to the destabilized state in Fig. 1.

Two examples of physiologically interacting/offsetting events which may occur as a result of changes in ambient air quality will be discussed using the concepts from the conceptual models of differential sensitivity (Fig. 1, 2).

Drought Stress--Pollutant Interactions

Drought stress induces a broad range of physiological and biochemical changes (e.g., Hsiao 1973, Hanson and Hitz 1982, Kramer 1983). For example, drought stress alters hormonal

levels, influences metabolic processes and enzyme activities, reduces carbon fixation, alters carbon allocation, reduces leaf conductance and H_2O potential and may increase leaf temperature. These metabolic and physiological changes ultimately influence plant growth and response to other stresses. Typically drought stress favors root growth at the expense of producing/maintaining leaf tissue or defensive compounds (Kramer 1983). This change in allocation can cause an additional metabolic readjustment or further decline and an increase in the susceptibility to pest attack or abiotic stresses (Schoeneweiss 1978). In contrast, drought-stressed plants are less susceptible to the herbicide paraquat than well-watered ones (Burke et al. 1985). In field grown Glycine max (L.) Merr., UV-B exposure reduced photosynthesis, transpiration, plant growth and number of pods (Murali and Teramura 1986), whereas, in drought-stressed plants, UV-B had no significant effects on these parameters. The drought-stressed plants had a higher specific leaf weight and methanolic extracts displayed a higher UV absorbance (300 nm).

Increased atmospheric CO_2 can alter plant response to H_2O deficits. The development of internal drought stress was delayed during a drying cycle in Liquidambar styraciflua L. grown at high CO_2 levels (Tolley and Strain 1984, 1985). Seedlings maintained higher photosynthetic rates during the drying cycle and exhibited increased H_2O use efficiency (WUE). The response may have a genetic basis since other species such as Pinus taeda L. did not

respond differentially to H_2O deficits when grown at high CO_2. Regardless of the mechanisms, increased WUE and drought tolerance may enable Liquidambar styraciflua L. to extend its range under elevated levels of CO_2.

Drought stress reduces the impacts of air pollutants on plants through both avoidance and tolerance mechanisms. Field and controlled environment studies have established that drought stressed plants display less foliar injury and O_3 impairs growth/yield to a lesser extent than in well-watered plants (e.g., Dean and Davis 1967, Markowski and Grzesiak 1974, Moser et al. 1988, Olszyk and Tibbitts 1981). The drought-induced reduction in stomatal conductance (an avoidance mechanism) reduces the uptake of gaseous pollutants such as O_3 (Rich and Turner 1972, Tingey and Hogsett 1985). Drought stress also increases plant resistance to oxidant stress through modification of gene expression. The levels of cellular antioxidant systems and glutathione reductase increase under water stress (Burke et al. 1985, Gamble and Burke 1984).

Queiroz (1988) suggested that drought stress perturbed the metabolic balance so that the energy and/or enzyme capacity required to repair or compensate for the additional stress from an air pollutant was no longer available. In a study of the interactive effects of drought stress and SO_2, a low concentration of SO_2 (80 PPB up to 6 weeks) had no effect on the

protein levels in well-watered Picea abies L. Karst. (5-month and 5-year old plants) (Pierre and Queiroz 1988, Queiroz 1988). Drought stress (H_2O withheld for 1 or 2 weeks) reduced foliar proteins 17 to 28%. The impact of drought stress was intensified by SO_2 which reduced the protein level by 38 to 54%. The activities of isocitrate, glucose-6-phosphate, and glutamate dehydrogenases showed the same response patterns as the protein levels. Foliar injury developed only on the plants that were drought stressed and exposed to SO_2. When the water-stressed plants were rewatered, the protein and enzyme activities increased to normal levels within a few days. However, in young Picea abies L. Karst. plants exposed to SO_2 and water stress, only a portion of the plants recovered (11% of the plants died). In another study, SO_2 had no effect on photosynthesis after 5 weeks in well watered Picea abies L. Karst. (Cornic 1987). When H_2O was withheld, reductions in photosynthesis were the greatest in SO_2 treated plants. Studies with field-grown Glycine max L. Merr. found that drought and O_3 stresses reduced yields 4 and 5%, respectively, while the combined stresses reduced yields 25% (Heggestad et al. 1985).

Interactions among various pollutants and natural stresses such as drought are complex. The above examples suggest a strong genetic basis for plant response. The examples clearly show that not all interactions will be detrimental. In fact, increasing the CO_2 concentration may ameliorate some of the detrimental

effects of gaseous pollutants and in some species may increase drought tolerance.

CO_2--Pollutant Interactions

Increasing atmospheric concentrations of CO_2 reduces stomatal conductance (e.g., Acock and Allen 1985), decreasing the uptake of other pollutants. Stomatal frequency decreased when the partial pressure and mole fraction of CO_2 increased (Woodward and Bazzaz 1988). However, stomatal frequency was unchanged when the partial pressure of CO_2 was held constant and mole fraction was increased, suggesting stomatal frequency was directly controlled by the CO_2 partial pressure. An examination of herbarium species collected from the 18th, 19th and 20th centuries supports the possibility that stomatal frequency has decreased as the ambient CO_2 concentrations have increased (Woodward 1987).

Despite effects of increased CO_2 on stomatal conductance and frequency, the overall effect of increased CO_2 on growth is stimulatory, particularly for C3 plants. Quercus alba L. seedlings grown under elevated CO_2 concentrations (690 ppm) showed greater growth and H_2O use efficiency than those grown under current ambient levels (Norby et al. 1986). The photosynthetic rates of plants grown at lower CO_2 concentrations (225 to 260 ppm compared to 340 ppm) were lower for a given stomatal frequency, suggesting that biochemical factors affecting CO_2 fixation were altered at the lower concentrations (Woodward

and Bazzaz 1988). Fetcher et al. (1988) found that elevated CO_2 increased growth of Pinus taeda L. and Liquidambar styraciflua L. even though carbon exchange rate (CER) of Liquidambar styraciflua L. was reduced. Further examination of the response revealed that the reduction in CER in Liquidambar styraciflua L. resulted from both stomatal limitations to CO_2 uptake and from a reduced biochemical capacity for CO_2 fixation, presumably from reduced ribulose bisphosphate carboxylase (rubisco) activity. The apparent conflict between an increase in net productivity and decreased CER in Liquidambar styraciflua L. may have resulted from a change in the CER:PPFD relationship, or from increased leaf area under elevated CO_2.

Because carbon fixation and growth is enhanced under elevated CO_2, additional substrates may be available for maintenance and repair processes, enabling the plant to tolerate higher levels of other pollutant stresses (Fig. 1, 2). For example, the inhibitory effects of $SO_2 + NO_2$ on the photosynthetic rate of Medicago sativa L. 'Ranger was 50% less at high CO_2 than at low CO_2 (Hou et al. 1977). The protection resulted from both avoidance of $SO_2 + NO_2$ uptake via stomatal closure at high CO_2, and increased inactivation of the toxic SO_3^{-2}. In Glycine max (L.) Merr., photosynthesis declined with SO_2 fumigation; the magnitude of the reduction was greater at low than high CO_2 levels (Carlson 1983). Further study showed that at equivalent SO_2 uptake rates, the reductions in photosynthesis

were less when the plants were grown at high CO_2. The results were similar to those of Carlson and Bazzaz (1982), which showed that high CO_2 protected plants via two mechanisms: stomatal closure increasing resistance to SO_2 uptake; and altered processes at the biochemical level. It is possible that increased carbohydrate was available for maintenance and repair processes, or for detoxification reactions. Koziol and Jordan (1978) found a correlation between photosynthesis and respiration rates, leaf carbohydrate levels, and pollutant damage in plants grown at ambient CO_2, and suggested that the availability of carbon substrates for maintenance and repair may be related to pollutant sensitivity.

Carlson and Bazzaz (1982) studied both C3 and C4 plants to determine the effects of elevated CO_2 on SO_2 toxicity. Growth of the C3 species was stimulated more than the growth of C4 species under elevated CO_2. At low CO_2, C3 plants were more sensitive to SO_2 than C4 plants, while at high CO_2, C4 plants were more sensitive to SO_2 than C3 plants. Differences in stomatal response between C3 and C4 species were suggested as factors controlling the differential response.

The overall effect of elevated CO_2 shifts the threshold concentration necessary to elicit a plant response to higher levels of SO_2 and/or NO_2. If elevated CO_2 decreases plant susceptibility via detoxification of toxic SO_3^{-2}, the activation

of specific enzyme systems may be involved. This would be an example of homeostatic readjustment through modification of gene expression (Fig. 1). Elevated CO_2 can provide the increased carbon substrate necessary for this metabolically expensive process, decreasing the susceptibility of a plant to an inciting stress (Fig. 2).

CONCLUSION

The conceptual model (Fig. 1) of plant response to pollutant stress is generic for various stresses regardless of their origin or site of action and has many features that are subject to genetic control. Plants have various mechanisms which enable them to 1) avoid (either through exclusion or detoxification systems), or 2) tolerate stress through metabolic readjustments. Metabolic readjustments involve utilization of reserve capacity with minimal energy cost, or modification of gene expression at relatively greater energy cost.

Traits affecting sensitivity to pollutants may be induced by other natural stresses. For example, water stress may induce the formation of oxidant scavengers which are also important in detoxifying O_3 and its derivatives. Consequently, it is important to distinguish between response to the pollutant and actual evolution of tolerance in response to selection by

pollutants.

The few studies which have examined the interactions of multiple stresses simultaneously have shown that plant responses are not additive. This may result in part from the complex nature of genotype/environment interactions and the role of environment on genotypic expression. Differential plant sensitivity is influenced by genetically determined traits mediated through various mechanisms.

Differential plant sensitivity is a result of a range of physiological processes which are subject to genetic control. The relative importance of tolerance and avoidance mechanisms in controlling sensitivity varies within and among species. The magnitude of the response is determined by the amount of the pollutant reaching the target site, and the ability of the plant to reestablish homeostatic equilibrium. In general, avoidance may be an effective accommodation to occasional acute episodes while tolerance is more likely with chronic pollutant exposure. However, mechanisms of response are dynamic and are influenced by a multitude of factors; tolerance and avoidance mechanisms likely do not act independently. Future research efforts should be directed at characterizing interactive roles of avoidance and tolerance processes and the interaction of various atmospheric stresses simultaneously rather than identifying responses to individual stresses.

REFERENCES

Abeles FB (1973) Ethylene in plant biology. Academic Press, New York

Acock B, Allen LH Jr. (1985) Crop responses to elevated carbon dioxide concentrations. In Strain BR, Cure JD (eds) Direct Effects of Increasing Carbon Dioxide on Vegetation, US Dept. of Energy, Office of Energy Research, Washington, DC, pp 53-97

Alscher R, Bower JL, Zipfel W (1987) The basis for different sensitivities of photosynthesis to SO_2 in two cultivars of pea. J. Exp. Bot. 38:99-108

Alscher RG, Amthor JS (1988) The physiology of free-radical scavenging: Maintenance and repair processes. In Schulte-Hostede S, Darrally NM, Blank LW, Wellburn AR (eds) Air pollution and Plant Metabolism, Elsevier Applied Science, N.Y.

Amthor JS (1988) Growth and maintenance respiration in leaves of bean (Phaseolus vulgaris L.) exposed to ozone in open-top chambers in the field. New Phytol. 100:319-325

Amthor JS, Cumming JR (1988) Low levels of ozone increase bean leaf maintenance respiration. Canadian J. Bot. 66:724-726

Ariens EJ, Simonis AM, Offermerier J (1976) Introduction to General Toxicology, Academic Press, New York

Ayazloo M, Garsed SG, Bell JNB (1982) Studies on the tolerance to sulfur dioxide of grass populations in polluted areas, II: Morphological and physiological investigations. New Phytol. 90:109-26

Barnes JD, Davison AW (1988) The Influence of ozone on the winter hardiness of Norway spruce [Picea abies (L.)]. New Phytol. 108:159-66

Barnes JD, Davison AW, Booth TA (1988) Ozone accelerates structural degradation of epicuticular wax on Norway spruce needles. New Phytol. 110:309-318

Barnes RL (1972) Effects of chronic exposure to ozone on photosynthesis and respiration of pines. Environ. Pollut. 3:133-138

Beggs CJ, Schneider-Ziebert U, Wellman E (1986) UV-B radiation and adaptive mechanisms in plants. In Worrest RC, Caldwell MM (eds) Stratospheric Ozone Reduction, Solar Radiation and Plant Life, Springer-Verlag, Berlin, pp 235-250

Beggs CJ, Stolzer-Jehle A, Wellmann E (1985) Isoflavonoid formation as an indicator of UV stress in bean (Phaseolus vulgaris L.) leaves. The significance of photorepair in assessing potential damage by increased solar UV-B radiation. Plant Physiol. 79:630-634

Bennett JH, Lee EH, Heggestad HE (1984) Biochemical aspects of plant tolerance to ozone and oxyradicals: Superoxide dismutase. In Koziol MJ, Whatley FR (eds) Gaseous Air Pollutants and Plant Metabolism, Butterworths, London, pp 413-424

Black CR, Black VJ (1979a) The effects of low concentrations of sulphur dioxide on stomatal conductance and epidermal cell survival in field bean (Vicia faba L.). J. Exp. Bot. 31:667-677

Black CR, Black VJ (1979b) Light and scanning electron microscopy of SO_2-induced injury to leaf surfaces of field bean (Vicia faba L.). Plant, Cell, and Env. 2:329-333

Black VJ, Unsworth MH (1980) Stomatal responses to sulfur dioxide and vapour pressure deficit. J. Exp. Bot. 31:667-677

Blakeley SD, Robaglia C, Brzezinski R, Thirion J-P (1986) Induction of low molecular weight cadmium-binding compound in soybean roots. J. Exp. Bot. 37(180):956-964

Bressan RA, LeCureux L, Wilson LG, Filner P (1979) Emission of ethylene and ethane by leaf tissue exposed to injurious concentrations of sulfur dioxide or bisulfite ion. Plant Physiol. 63:924-930

Bressan RA, LeCureux L, Wilson LG, Filner P, Baker LR (1981) Inheritance of resistance to sulfur dioxide in cucumber. HortScience 16:332-333

Burke JJ, Gamble PE, Hatfield JL, Quisenberry JE (1985) Plant morphological and biochemical responses to field water deficits. I. Responses of glutathione reductase activity and paraquat sensitivity. Plant Physiol. 79:415-419

Butler LK, Tibbitts TW, Bliss FA (1979) Inheritance of resistance to ozone in Phaseolus vulgaris L. J. Am. Soc. Hort. Sci. 104:211-213

Caldwell MM, Robberecht R, Flint SD (1983) Internal filters: Prospects for UV-acclimation in higher plants. Physiol. Plant. 5:445-450

Carlson RW (1983) The effect of SO_2 on photosynthesis and leaf resistance at varying concentrations of CO_2. Environ. Pollut. (Series A) 30:309-321

Carlson RW, Bazzaz FA (1982) Photosynthetic and growth response to fumigation with SO_2 at elevated CO_2 for C_3 and C_4 plants. Oecologia 54:50-54

Castillo FJ, Greppin H (1986) Balance between anionic and cationic extracellular peroxidase activities in Sedum album leaves after ozone exposure. Analysis by high-performance liquid chromatography. Physiol. Plant. 68:201-208

Chameides WL (1989) The chemistry of ozone deposition to plant leaves: Role of ascorbic acid. Environ. Sci. Technol. 23:595-600

Chanway CP, Runeckles VC (1984a) The role of superoxide dismutase in the susceptibility of bean leaves to ozone injury. Can. J. Bot. 62:236-240

Chanway CP, Runeckles VC (1984b) Effect of ethylene diurea (EDU) on ozone tolerance and superoxide dismutase activity in bush bean. Environ. Pollut. (Series A) 35:49-56

Chappell J, Hahlbrock K (1984) Transcription of plant defense genes in response to UV-light or fungal elicitor. Nature 311:76-78

Cherian MG, Goyer RA (1978) Metallothioneins and their role in the metabolism and toxicity of metals. Life Sciences 23(1):1-10

Cornic G (1987) Interaction between a sublethal pollution by SO_2 and water stress. The effect on photosynthetic capacity. Physiol. Plant. 71:115-119

DeCormis L (1968) Dégagement d'hydrogène sulfuré par des plantes soumises à une atmosphère contenant de l'anhydride sulfureux. Compte rendu de l'Academie de Sciences (serie D) 266:683-85

Dean CR, Davis DR (1967) Ozone and soil moisture in relation to the occurrence of weather fleck on Florida cigar-wrapper tobacco in 1966. Plant Dis. Rep. 51:72-75

Dodrill SA (1976) Fate of Sulfur-Dioxide Absorbed by Foliage of Coleus and Bean in Relation to Visible Injury. Ph.D. Thesis, Univ. of West Virginia

Dragoescu N, Hill RR Jr., Pell EJ (1988) An autotetraploid model for genetic analysis of ozone tolerance in potato, Solanum tuberosum L. Genome 29:85-90

Engle RL, Gabelman WH (1966) Inheritance and mechanism for resistance to ozone damage in onion, Allium cepa L. Proc. Am. Soc. Hortic. Sci. 89:423-430

Fetcher N, Jaeger CH, Strain BR, Sionit N (1988) Long-term elevation of atmospheric CO_2 concentration and the carbon exchange rates of saplings on Pinus taeda L. and Liquidambar styraciflua L. Tree Physiol. 4:255-262

Fridovich I (1978) The biology of oxygen radicals. Science 201:875-880

Gamble PE, Burke JJ (1984) Effect of water stress on the chloroplast antioxidant system. I. Alterations in glutathione reductase activity. Plant Physiol. 76:615-621

Grill E, Winnacker E-L, Zenk MH (1985) Phytochelatins: The principal heavy-metal complexing peptides of higher plants. Science 230:674-676

Grünhage L, Weigel H-J, Ilge D, Jäger H-J (1985) Isolation and partial characterization of a cadmium-binding protein from Pisum sativum. Plant Physiol. 119:327-334

Gunderson CA, Taylor GE Jr. (1988) Kinetics of inhibition of foliar gas exchange by exogenous ethylene: An ultrasensitive response. New Phytol. 110:517-524

Hällgren JE, Fredricksson SA (1982) Emission of hydrogen sulfide from sulfur dioxide fumigated pine trees. Plant Physiol. 70:456-459

Hanson AD, Hitz WD (1982) Metabolic responses of mesophytes to plant water deficits. Ann. Rev. Plant Physiol. 33:163-203

Hanson GP, Addis DH, Thorne L (1976) Inheritance of photochemical air pollution tolerance in petunia. Can. J. Genet. and Cytol. 6:75-83

Hanson GP, Thorne L, Jativa CD (1971) Ozone tolerance of petunia leaves as related to their ascorbic acid concentrations. In Englund HM, Beery WT (eds) Proceedings of the Second International Clean Air Congress, Academic Press, New York, pp 261-266

Heath RL (1980) Initial events in injury to plants by air pollutants. Ann. Rev. Plant Physiol. 31:395-431

Heath RL, Chimiklis P, Frederick P (1974) The role of Potassium and lipids in ozone injury to plant membranes. In Dugger M (ed) ACS Symposium Series 3, American Chemical Society, Washington, DC, pp 58-75

Heggestad HE, Gish TJ, Lee EH, Bennett JH, Douglass LW (1985) Interaction of soil moisture stress and ambient ozone on growth and yields of soybeans. Phytopathology 75:472-477

Hou L-Y, Hill AC, Soleimani A (1977) Influence of CO_2 on the effects of SO_2 and NO_2 on alfalfa. Environ. Pollut. 12:7-16

Houston DB, Stairs GR (1973) Genetic control of sulfur dioxide and ozone tolerance in eastern white pine. For. Sci. 19:267-271

Howe TK, Woltz SS (1981) Resistance of tomato cultivars to sulfur dioxide and accumulation of foliar sulfite related to sulfur dioxide susceptibility. HortScience 16:413

Howell RK, Kremer DF (1973) The chemistry and physiology of pigment in leaves injured by air pollution. J. Environ. Qual. 2:434-438

Hsiao TC (1973) Plant responses to water stress. Ann. Rev. Plant Physiol. 24:519-570

Johnson AH (1989) Decline of red spruce in the northern Applachians: Determining if air pollution is an important factor. In Biologic Markers of Air Pollution Stress and Damage in Forests, National Academy Press, Washington, DC, pp 91-104

Karnosky DF (1977) Evidence for genetic control of response to sulfur dioxide and ozone in Populus tremuloides. Can. J. For. Res. 7:437-440

Karnosky DF, Berrang PC, Scholz F, Bennett JP (1989) Variation in and natural selection for air pollution tolerances in trees. In Scholz F, Gregorius H-R, Rudin D (eds) Genetic Effects of Air Pollutants in Forest Tree Populations, Springer Verlag, Berlin

Kerstiens G, Lendzian KJ (1989) Interactions between ozone and plant cuticles. I. Ozone deposition and permeability. New Phytol. 112:13-19

Kondo N, Akiyama Y, Fujiwara M, Sugahara K (1980) Sulfite-oxidizing activities in plants. In Studies on the Effects of Air Pollutants on Plants and Mechanisms of Phytotoxicity, Research Report No. 11 for the National Institute of Environmental Studies, Iburaki, Japan, pp 137-150

Koukol J, Dugger WM Jr. (1967) Anthocyanin formation as a response to ozone and smog treatment in Rumex crispus L. Plant Physiol. 42:1023-1024

Koziol MJ, Jordan CF (1978) Changes in carbohydrate levels in red kindey bean (Phaseolus vulgaris L.) exposed to sulphur dioxide. J. Exp. Bot. 29(112)1037:1043

Kramer PJ (1983) Water Relations of Plants. Academic Press, New York

Kuhn DN, Chappel J, Boudet A, Hahlbrock K (1984) Induction of phenylahanine ammonia-lyase and 4-coumarate:CoA ligase mRNAs in cultured plant cells by UV light or fungal elicitor. Proc. Natl. Acad. Sci. USA 81:1102-1106

Larcher W (1975) Physiological Plant Ecology. Springer-Verlag, Berlin, Heidelberg, New York

Lee EH, Bennett JH (1982) Superoxide dismutase. A possible protective enzyme against ozone injury in snap beans (Phaseolus vulgaris L.). Plant Physiol. 69:1444-1449

Lee EH, Jersey A, Gifford C, Bennett J (1984) Differential ozone tolerance of soybean and snapbeans: Analysis of ascorbic acid in O3-susceptible and O3-resistant cultivars by high-performance liquid chromatography. Environ. Exp. Bot. 24:331-341

Leshem YY (1988) Plant senescence processes and free radicals. Free Radical Biology & Medicine 5:39-49

Levitt J (1972) Responses of Plants to Environmental Stress. Academic Press, New York

Lindberg SE, Lovett GM, Richter DD, Johnson DW (1986) Atmospheric deposition and canopy interactions of major ions in a forest. Science 231:143-145

Manion PD (1981) Tree Disease Concepts. Prentice Hall, New Jersey

Markowski A, Grzesiak S (1974) Influence of sulphur dioxide and ozone on vegetation of bean and barley plants under different soil moisture conditions. Bulletin L'Académie Polonaise Sciences Série sciences biologiques 22:875-887

Matters GL, Scandalios JG (1986) Changes in plant gene expression during stress. Developmental Genetics 7:167-175

McKersie BD, Beversdorf WD, Hucl P (1982) The relationship between ozone insensitivity, lipid soluble antioxidants, and superoxide dismutase in Phaseolus vulgaris. Can. J. Bot. 60:2686-2691

McLaughlin SB, Taylor GE (1981) Relative humidity: Important modifier of pollutant uptake by plants. Science 211:167-168

Miller JE, Xerikos P (1979) Residence time of sulfite in SO_2 "sensitive" and "tolerant" soybean cultivars. Environ. Pollut. 18:259-64

Mirecki RM, Teramura AH (1984) Effects of ultraviolet-B irradiance on soybean. V. The dependence of plant sensitivity on the photosynthetic photon flux density during and after leaf expansion. Plant Physiol. 74:475-480

Moser TJ, Tingey DT, Rodecap KD, Rossi DJ, Clark CS (1988) Drought stress applied during the reproductive phase reduced ozone-induced effects in bush bean. In Heck WW, Taylor OC, Tingey DT (eds) Assessment of Crop Loss from Air Pollutants, Elsevier Applied Science, London, pp 345-364

Mudd JB (1982) Effects of oxidants on metabolic function. In Unsworth MH, Ormrod DP (eds) Effects of Gaseous Air Pollution in Agriculture and Horticulture, Butterworths, London, pp 189-203

Murali NS, Teramura AH (1986) Effectiveness of UV-B radiation on the growth and physiology of field-grown soybean modified by water stress. Photochem. and Photobiol. 44(2):215-219

Noble PS (1974) Biophysical Plant Physiology. W.H. Freeman and Company, San Francisco

Norby RJ, O'Neill EG, Luxmoore RJ (1986) Effects of atmospheric CO_2 enrichment on the growth and mineral nutrition of Quercus alba seedlings in nutrient-poor soil. Plant Physiol. 82:83-89

Norby RJ, Weerasuriya Y, Hanson PJ (1989) Induction of nitrate reductase activity in red spruce needles by NO_2 and HNO_3 vapor. Can. J. For. Res. 19:889-896

Olszyk DM, Tibbitts TW (1981) Stomatal response and leaf injury of Pisum sativum with SO_2 and O_3 exposures. II. Influence of moisture stress and time of exposure. Plant Physiol. 67:545-549

Peiser G, Yang SF (1985) Biochemical and physiological effects of SO_2 on nonphotosynthetic processes in plants. In Winner WE, Mooney HA, Goldstein RA (eds) Sulfur Dioxide and Vegetation, Physiology, Ecology, and Policy Issues pp 148-161

Pell EJ (1974) The impact of ozone on the bioenergetics of plant systems. In Dugger M (ed) Air Pollution Effects on Plant Growth, ACS Symposium Series 3, American Chemical Society, Washington, DC, pp 106-114

Pierre M, Queiroz O (1988) Air pollution by SO_2 amplifies the effects of water stress on enzymes and total soluble proteins of spruce needles. Physiol. Plant. 73:412-417

Pitelka LF (1988) Evolutionary responses of plants to anthropogenic pollutants. Trends in Ecology & Evolution 3:233-236

Queiroz O (1988) Air pollution, gene expression and post-translational enzyme modifications. In Schulte-Hostede S, Darrall NM, Blank LW, Wellburn AR (eds) Air Pollution and Plant Metabolism, Elsevier Applied Science, London, pp 238-254

Raddi P, Rinallo C (1989) Variation in needle wax degradation in two silver fir provenances differentiated by tolerance to air pollution. In Scholz F, Gregorius H-R, Rudin D (eds) Genetic Effects of Air Pollutants in Forest Tree Populations, Springer Verlag, Berlin

Rauser WE (1984) Isolation and partial purification of cadmium-binding protein from roots of the grass Agrostis gigantea. Plant Physiol. 74:1025-1029

Rauser WE, Curvetto NR (1980) Matellothionein occurs in roots of Agrostis tolerant to excess copper. Nature 287:563-564

Reich PB (1987) Quantifying plant response to ozone: A unifying theory. Tree Physiol. 3:63-91

Rennenberg H (1982) Glutathione metabolism and possible biological roles in higher plants. Phytochemistry 21(22):2771-2781

Rennenberg H (1984) The fate of excess sulfur in higher plants. Ann. Rev. Plant. Physiol. 35:121-53

Rich S, Turner NC (1972) Importance of moisture on stomatal behavior of plants subjected to ozone. J. Air Pollut. Cont. Assoc. 22:718-721

Robberecht R, Caldwell MM (1986) Leaf UV optical properties of Rumex patientia L. and Rumex obtusifolius L. in regard to a protective mechanism against solar UV-B radiation injury. In Worrest RC, Caldwell MM (eds) Stratospheric Ozone Reduction, Solar Radiation and Plant Life, Springer-Verlag, Berlin, pp 251-259

Robinson NJ, Jackson PJ (1986) "Metallothionein-like" metal complexes in angiosperms; their structure and function. Physiol. Plant. 67:499-506

Rogers HH, Campbell JC, Volk RJ (1979) Nitrogen-15 Dioxide uptake and incorporation by Phaseolus vulgaris (L.). Science 206:333-335

Roose ML, Bradshaw AD (1982) Evolution of resistance to gaseous air pollutants. In Unsworth MH, Ormrod DP (eds) Effects of Gaseous Air Pollution in Agriculture and Horticulture, Butterworth Scientific, London, pp 379-409

Rowland AJ (1986) Nitrogen uptake, assimilation and transport in barley in the presence of atmospheric nitrogen dioxide. Plant and Soil 91:353-356

Rowland AJ, Drew MC, Wellburn AR (1987) Foliar entry and incorporation of atmospheric nitrogen dioxide into barley plants of different nitrogen status. New Phytol. 107:357-371

Sachs MM, Ho T-HD (1986) Alteration of gene expression during environmental stress in plants. Ann. Rev. Plant Physiol. 37:363-376

Sage RF, Sharkey TD, Seemann JR (1989) Acclimation of photosynthesis to elevated CO_2 in five C_3 species. Plant Physiol. 89:590-596

Salin ML (1988) Toxic oxygen species and protective systems of the chloroplast. Physiol. Plant. 72:681-689

Sauter JJ, Voss JV (1986) SEM-observations on the structural degradation of epistomatal waxes in Picea abies (L.) Karst. and its possible role in the "Fichtensterben". Eur. J. For. Path. 16:408-423

Scandalios JG (1987) The antioxidant enzyme genes Cat and Sod of maize: Regulation, functional significance, and molecular biology. Current Topics in Biological and Medical Research 14:19-44

Schoeneweiss DF (1978) Water stress as a predisposing factor in plant disease. In Kozlowski TT (ed) Water deficits and plant growth, Academic Press, New York, 5:61-99

Scholz F, Gregorius H-R, Rudin D (eds) (1989) Genetic Effects of Air Pollutants in Forest Tree Populations, Springer Verlag Berlin, Heidelberg, New York, pp 104

Silvius JE, Baer CH, Dodrill S, Patrick H (1976) Photoreduction of sulfur dioxide by spinach leaves and isolated spinach choloroplasts. Plant Physiol. 57:799-801

Skärby L, Troeng E, Boström C-A (1987) Ozone uptake and effects on transpiration, net photosynthesis, and dark respiration in Scots pine. For. Sci. 33(3):801-808

Steinmüller D, Tevini M (1986) UV-B-induced effects upon cuticular waxes of cucumber, bean and barley leaves. In Worrest RC, Caldwell MM (eds) Stratospheric Ozone Reduction, Solar Radiation and Plant Life, Springer-Verlag, Berlin, pp 261-269

Sutton R, Ting IP (1977a) Evidence of repair of ozone induced membrane injury; alteration in sugar uptake. Atmos. Environ. 11:273-275

Sutton R, Ting IP (1977b) Evidence for the repair of ozone induced membrane injury. Am. J. Botany 64:404-411

Tanaka K, Suda Y, Kondo N, Sugahara K (1985) O_3 tolerance and the ascorbate-dependent H_2O_2 decomposing system in chloroplasts. Plant Cell Physiol. 26:1425-1431

Taylor GE Jr. (1978) Genetic analysis of ecotypic differentiation within an annual plant species, Geranium carolinianum L. in response to sulfur dioxide. Bot. Gaz. 139:362-368

Taylor GE Jr., Tingey DT (1983) Sulfur dioxide flux into leaves of Geranium carolinianum L.: Evidence for a nonstomatal or residual resistance. Plant Physiol. 72:237-244

Taylor GE Jr., Tingey DT, Gunderson CA (1986) Photosynthesis, carbon allocation, and growth of sulfur dioxide ecotypes of Geranium carolinianum L. Oecologia 68:350-357

Thomas MD (1961) Effects of air pollution on plants. In World Health Organization, Air Pollution, New York: Columbia University Press, pp 233-278

Thorne L, Hanson GP (1972) Species differences in rates of vegetal ozone absorption. Environ. Pollut. 3:303-312

Thorne L, Hanson GP (1976) Relationship between genetically controlled ozone sensitivity and gas exchange rate in Petunia hybrida Vilm. J. Am. Soc. Hort. Sci. 101:6-63

Tingey DT (1980) Stress ethylene production - A measure of plant response to stress. HortScience 15(5):630-633

Tingey DT, Fites RC, Wickliff C (1976) Differential foliar sensitivity of soybean cultivars to ozone associated with differential enzyme activities. Physiol. Plant. 37:69-72

Tingey DT, Hogsett WE (1985) Water stress reduces ozone injury via a stomatal mechanism. Plant Physiol. 77:944-947

Tingey DT, Olszyk DM (1985) Intraspecific variability in metabolic responses to SO_2. In Winner WE, Mooney HA, Goldstein RA (eds) Sulfur Dioxide and Vegetation, Stanford University Press, Stanford, CA, pp 178-205

Tingey DT, Taylor GE (1982) Variation in plant response to ozone: A conceptual model of physiological events. In Unsworth MH, Ormrod DP (eds) Effects of Gaseous Air Pollution in Agriculture and Horticulture, Butterworth Scientific, London, pp 113-138

Tingey DT, Thutt GL, Gumpertz ML, Hogsett WE (1982) Plant water status influences ozone sensitivity of bean plants. Agric. and Environ. 7:243-254

Tolley LC, Strain BR (1984) Effects of CO_2 enrichment and water stress on growth of Liquidambar styraciflua and Pinus taeda seedlings. Can. J. Bot. 62:2135-2139

Tolley LC, Strain BR (1985) Effects of CO_2 enrichment and water stress on gas exchange of Liquidambar styraciflua and Pinus taeda seedlings grown under different irradiance levels. Oecologia 65:166-172

Wagner GJ, Trotter MM (1982) Inducible Cadmium Binding Complexes of Cabbage and Tobacco. Plant Physiol. 69:804-809

Warner CW, Caldwell MM (1983) Influence of photon flux density in the 400-700 nm waveband on inhibition of photosynthesis by UV-B (280-320 nm) irradiation in soybean leaves: Separation of indirect and immediate effects. Photochem. and Photobiol. 38(3):341-346

Wellburn AR (1982) Effects of SO_2 and NO_2 on metabolic function. In Unsworth MH, Ormrod DP (eds) Effects of Gaseous Air Pollution in Agriculture and Horticulture, Butterworths, London, pp 169-187

Wellburn AR, Majernik O, Wellburn F (1972) Effects of SO_2 and NO_2 polluted air upon the ultrastructure of chloroplasts. Environ. Pollut. 3:37-49

Winner WE, Gillespie C, Shen W-S, Mooney HA (1988) Stomatal responses to SO_2 and O_3. In Schulte-Hostede S, Darrall NM, Blank LW, Wellburn AR (eds) Air Pollution and Plant Metabolism, Elsevier Applied Science Publishers LTD, England, pp 255-271

Woodward FI (1987) Stomatal numbers are sensitive to increases in CO_2, from pre-industrial levels. Nature 327:617-618

Woodward FI, Bazzaz FA (1988) The responses of stomatal density to CO_2 partial pressure. J. Exp. Bot. 39(209):1771-1781

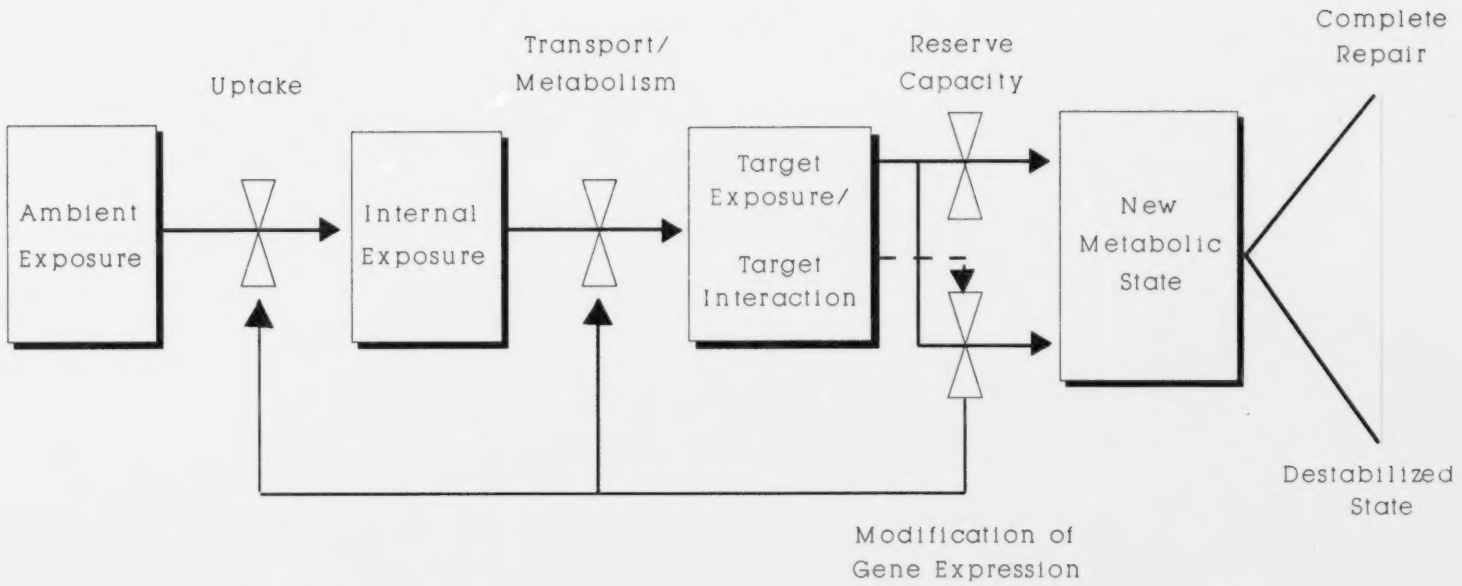
Zeevaart AJ (1974) Induction of nitrate reductase by NO_2. Acta Bot. Neerl. 23:345-346

Zeevaart AJ (1976) Some effects of fumigating plants for short periods with NO_2. Environ. Pollut. 11:97-100

FIGURE LEGENDS

Fig. 1. Conceptual model of the processes controlling differential sensitivity to pollutants. The pollutant concentration will decrease as it moves from the ambient exposure to the target site. The generalized avoidance and tolerance processes are represented as values. Avoidance processes control the amount of pollutant at the target site and tolerance processes control the plant response (repair/compensation) to the stress. The response may also be a feedback process that influences the avoidance mechanisms.

Fig. 2. Conceptual model of energy (carbon) available to the plant for avoidance and repair/compensation processes (Adapted from Johnson, 1989). See text for details.


Ambient Exposure
Uptake
Internal Exposure
Transport/ Metabolism
Target Exposure/ Target Interaction
Reserve Capacity
New Metabolic State
Complete Repair
Destabilized State
Modification of Gene Expression

CARBON AVAILABLE FOR DEFENSE AND REPAIR